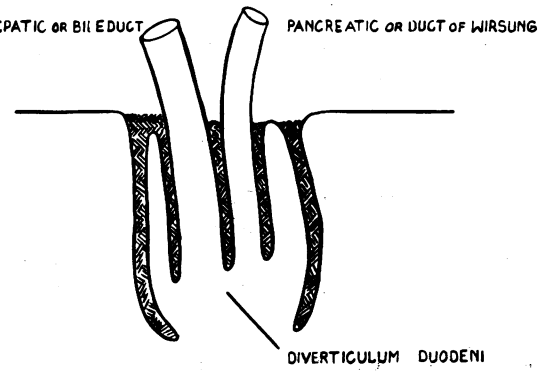
Pancreatitis of the Horse

By C. M. HIGGINSON*

PANCREATITIS in the horse is rarely mentioned in veterinary literature. But this disease must be more common than is generally supposed since the author has seen five cases within recent years, each of them confirmed by necropsy. Because each presented symptoms of abdominal disease together with variable other symptoms it is easy to visualize how a correct interpretation of symptoms was overlooked, especially before attention was drawn to the lesions by post mortem examination.

Before considering the material to be presented, a few salient points of the physiology and particularly the anatomy of the pancreatic gland of the horse should be borne in mind. The pancreatic gland is racemose and resembles the salivary gland. The cells lining the alveoli furnish the ferments. The secretion is collected by a branching system of ducts which form two radicles, draining the right and left side and ending in a principal duct — duct of Wirsung — which together with the bile duct pierce the intestine at a point about six inches from the pylorus and form a common orifice known as diverticulum duodeni. An accessory or small duct arises either from the duct of Wirsung or its left radicle and pierces the duodenum at a point opposite the diverticulum duodeni. Thus we see that the principal drainage of the gland is the duct of Wirsung. If this is obstructed at the diverticulum, the accessory duct in some animals may compensate, but in others where the accessory takes its origin from the left branch, this is impossible



The pancreatic juice contains three pricipal ferments — trypsin, amylopsin and lipase. Trypsin in the gland possesses little or no enzyme properties. It is only when it enters the intestine and is activated by the enterokinase found there that its powerful proteolytic property is developed.

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Enterokinase is not, however, the only substance which activates trypsin; in fact, body tissues generally appear to possess this property.

Amylopsin converts starch but need not be considered further since it has no bearing on the subject under discussion.

Lipase has the property of splitting fat. When the gland is damaged, permitting the escape of this enzyme into the tissues, it also plays a part in the pathological picture of pancreatitis.

It is stated by Chiari that all stages of pancreatic damage is brought about by the digestive action of trypsin, i.e. that one of the ferments secreted by the gland becomes activated and attacks it, thus bringing about the pathological change. This cannot take place under normal conditions in that trypsin alone possesses no digestive properties, but is dependent for activation upon succus entericus or other substances. Thus the digestive action of this ferment within the secreting gland is predicated upon its being activated *within the gland* by a substance such as enterokinase, bile, free blood plasma, or tissue cells.

From this it is supposed that the activation results from one or more of four primary conditions:—(1)

(a) Entrance of bile into pancreatic ducts.
(b) Obstruction to the free flow of pancreatic secretion.
(c) Thrombosis of venous radicles of gland.
(d) Mechanical trauma such as wounds.

It should be mentioned that the first two conditions may result from bacterial invasion of one or both of the ducts or the bowel at a point contiguous to the ducts' outlet. Thus in this sense the local bacterial invasion, innocent in itself, may be considered as the excitant of a train of pathological changes which results in death.

For years, many believed that the common cause of pancreatitis — in animals other than the horse — was an occlusion of the ampulla of Vator (diverticulum duodeni) by biliary calculi, thus preventing the flow of bile, damming it back into the pancreatic duct and activating the available trypsin. Some experimental support of this view was afforded by the demonstration that injections of sterile bile into pancreatic ducts were followed by pancreatitis. With this conception of the origin of pancreatitis, it is apparent why horses were considered unlikely to be affected, especially having regard to the absence of a gall bladder and a consequent absence of gall stones.

Recently the excellent experimental study of Rich and Duff (2) has added new knowledge and a new conception of the pathology of pancreatitis. Substantial evidence is presented that in man what they term, "metaplasia of the duct epithelium" is the cause of the majority of cases. In short, it is demonstrated that the epithelium of affected areas of the duct changes from the normal cuboidal or columnar to transitional or basal, and that this new type of epithelium leads to the formations of masses of cells

which block the duct. The ductules and acini behind the obstruction become dilated and the distended walls ruptured, thus permitting an escape of the pancreatic juice into the glandular substance. Interstitial pancreatic tissue, in common with other tissues of the body, has the power of activating trypsin. Therefore the trypsin of the escaped juice is activated and brings about in the affected areas digestion of the blood vessels' walls. This, together with the digestion of fat by the liberated lipase, completes the pathological picture. It will be noted that although there are different explanations of the methods by which obstruction takes place, still there is agreement that the major damage is done by activated trypsin.

A point of interest is an unusual manifestation which horses show to the absorption of toxic substances. All practitioners have repeatedly noted that metritis and certain types of digestive disturbances are frequent forerunners of laminitis. Apparently the toxic substances are absorbed and carried by the circulation to the capillaries of the laminae which are especially susceptible. An inflammatory process is thereby set up. It is significant that three of the five horses mentioned in this report, and affected with pancreatitis, also developed laminitis.

Case 1

An imported percheron stallion, five years of age, came under observation. Being imported, his history was only known for approximately a year but during this time he had always appeared healthy.

Present illness:—When first seen, he was showing the usual symptoms of laminitis, together with an obstinate constipation and mild abdominal pain. Requisite treatment was prescribed and daily examinations were made over a period of five days. Since at this time the laminitis had disappeared it was expected that an elevation of temperature (103°F.) which still remained would soon subside. The attendant was instructed to take morning and evening temperatures and report in the course of a few days if a normal level was not reached. No news was received until sixteen days later, at which time the intestinal symptoms had increased in severity and the attendant had become alarmed.

It was then found that the temperature had been taken twice daily during the sixteen days and at no time registered below 103°F. Food had been refused for several days. Laxatives failed to relieve a severe, persistent constipation. Clay coloured faeces, covered with a mucous coat resembling asbestos, and a scanty urine, bright yellow in colour, were present. The temperature registered *104.6°F.*, pulse *96*, and respirations *24*.

Physical Examination

1. *Head*

Mucous membrane of the eyes—slightly yellow.
No nasal discharge.
Mucous membrane of mouth and tongue—icteric.

2. *Chest*

The respiratory movement, although rapid, was normal in character and equal on both sides. Percussion and auscultation failed to reveal evidence of pneumonia or pleurisy.

3. *Abdomen*

The animal's behaviour evidenced dull abdominal pain, but with no gaseous distention. A rectal examination revealed a well loaded colon. The abdominal wall showed no rigidity. An examination failed to disclose hernia, and the testicles were normal in size with no tenderness.

4. *Legs and extremities*

The laminitis present at the first examination had disappeared and the animal showed no pain when moved. The opposite legs were equal in size and no corded lymphatics were discernible.

Because of the condition of the faeces, the icteric mucous membranes, and the abdominal disturbance, the condition was tentatively diagnosed jaundice, thought to be probably due to a low grade infection in the duodenum. (The required treatment for this condition was prescribed.)

For the next seventeen days little change in the condition appeared except that the temperature became somewhat higher, and rarely registered below 104°F. Laxatives or enemas failed to overcome the obstinate constipation. In spite of forcing liquids the animal rapidly lost weight.

At this time the jaundice became more pronounced and all visible mucous membranes took on an intense yellow appearance. Small petechia appeared on the mucous membrane of the eye. The temperature rose to 105.4°F., sweating became severe and in a few hours the animal went into coma, dying three days later.

Necropsy

Examination was undertaken immediately following death. All tissues were intensely bile-stained. Excepting the liver and pancreas, all organs otherwise appeared normal.

Bright yellow in colour, the liver was enlarged, and weighed over thirty pounds. The hepatic duct was enlarged, filled with bile, and obstructed at the duodenal opening. The bile contents could not be squeezed through the diverticulum. The major portion of the pancreas was necrotic, with the few remaining normal areas studded with petechia. The duct of Wirsung was enlarged and filled with necrotic material, the orifice of the duct being occluded. This with the occlusion of the bile duct appeared to have resulted from a local area of inflammation, which involved a few inches of the intestine in the region of the diverticulum duodeni.

Case 2.

A previously healthy Clydesdale gelding, seven years old, developed an obstinate constipation. Treated for three weeks with domestic remedies which gave only partial relief, other symptoms became manifest and the animal was placed under professional care.

At the time of first examination, the temperature registered *106°F.*, pulse *104*, and respirations *32*.

Physical Examination

1. *Head.*

Mucous membranes of the eyes—stained yellow.

No nasal discharge, but nasal mucous membranes yellow and suffused with blood.

Mouth contained a tenacious mucus with mucous membranes stained yellow.

2. *Chest.*

Respiratory excursions, although rapid, equal on both sides.

Auscultation and percussion failed to disclose evidence of pneumonia and pleurisy.

3. *Abdomen.*

Animal gave evidence of pain referable to the abdomen.

Distention could not be found, nor were abdominal walls rigid.

Rectal examination failed to disclose an overloaded bowel, but faeces putty colored and covered with mucus resembling asbestos.

4. *Extremities.*

Animal showed great reluctance to move, and when forced to do so evidenced pain.

Movements were stiff and the legs held under the body, giving a picture of laminitis, involving all four feet. Lymphangitis did not evidence itself.

The illness was thought to be jaundice with an accompanying laminitis, with management and treatment planned accordingly.

The progress of the disease was rapidly downward, jaundice becoming more marked and emaciation unbelievably rapid. On the sixth day after first examination the animal became comatose and died the following day.

Necropsy

Post mortem examination commenced a few minutes after death. The tissues were not as deeply bile stained as the mucous membranes would suggest. The principal lesion was pancreatic damage. In contrast to Case 1, no soft necrosis was found. The pancreas appeared atrophied, being less than one half normal size. Section disclosed, throughout the organ, many caseous areas about one inch in diameter. These were not surrounded by a fibrous capsule. An enlarged duct contained cheese-like material. The liver was enlarged, its duct distended with bile, which required considerable pressure to force into the bowel. A section of the intestine for about fifteen inches from the pylorus was inflamed.

Case III.

A Belgium stallion, six years of age, had suffered an attack of influenza a year before, recovering without interruption. With this exception, he had always been in excellent health and condition.

The illness referred to in this report commenced five weeks before examination. The animal was said to first show abdominal pain associated with a stubborn constipation which was not relieved by laxatives.

When first examined, the temperature registered *104.3°F.*, pulse *92*, and respirations *40*. Exhibiting a staggery gait, the animal appeared to have difficulty in maintaining his balance. The body was wet with perspiration.

Physical Examination

1. *Head.*

Mucous membranes of the eye, yellow and studded with small petechia.
Nostrils dilated and a slight mucous discharge came from the left side.
Visible mucous membranes icteric but no haemorrhages present.
Mouth contained a tenacious mucus, with mucous membranes of mouth and tongue yellow.

2. *Thorax*

Respiratory movements, although laboured, uniform on each side.
An examination of the chest disclosed no evidence of pneumonia or pleurisy.
Oedema present in the tissues along the sternum, which was cool, painless and pitted on pressure.
Faint heart sounds suggested myocarditis.

3. *Abdomen.*

Marked pains, referable to the abdomen, evidenced, but not caused by gaseous distention or an overloaded bowel.
Constipation marked and the voided faeces hard, putty colored, and covered with an asbestos-like mucus.
Some muscle rigidity noted.
Peristaltic sounds very sluggish.

4. *Extremities.*

Legs normal in size with no enlarged lymphatics.
Cardinal symptoms of laminitis, involving the two fore feet, presented themselves.

Diagnosis

Having regard to the preceding cases a diagnosis of pancreatitis with involvement of the bowel duct was made.

The animal grew gradually worse, emaciation being remarkably rapid. Nine days following the first examination death occurred.

Necropsy

The small intestine appeared normal but the diverticulum duodeni was filled with a firm, hard concretion. The bile duct was distended and the liver slightly enlarged.

A very enlarged pancreas, on section, showed blood extravasated into its tissue. Many small petechia present. The duct although enlarged presented no visible obstruction which would interfere with the flow of pancreatic juice except the concretion in the diverticulum.

Case IV.

A grade mare, nine years of age and weighing 1500 pounds, had been purchased in the open market about a year previous to the onset of illness. Therefore, her available history covered only a short period of time. The first symptoms appeared three months before examination. She had repeated attacks of so-called "colic", and for three months her appetite had been poor. She perspired freely. When first examined, temperature registered *105.2°F.*, pulse *88*, and respirations *31*. The gait was staggery, and

when quiet she inclined her neck and pushed the head with force against any nearby object.

Physical Examination

1. *Head.*

Mucous membranes of eyes, icteric, with small petechia present.

No discharge, but when nostrils were viewed with a light, mucous membranes found to be stained yellow.

Mucous membranes of tongue and mouth, yellow in appearance and coated with a thick tenacious mucus.

2. *Chest.*

Respiratory movements equal on both sides, and auscultation and percussion revealed nothing abnormal in the lungs or pleura.

Excepting slight dullness, heart sounds normal.

3. *Abdomen.*

Abdomen appeared somewhat distended. Rectal examination revealed an impacted colon and caecum.

Faeces, clay coloured, and covered with thick tenacious mucus.

3. *Extremities.*

No laminitis found. The left rear leg presented corded lymphatics which were painful to pressure.

Medication aimed at controlling any inflammatory process which might be present in the small intestine and general supportive treatment, including saline injections, was administered. The animal became gradually worse and died eight days following first examination.

Necropsy

Although the hepatic duct was distended with bile and the orifice apparently occluded, the liver presented a normal appearance with the exception of an increase in size.

The pancreas was very enlarged, and free blood found in many areas, more especially in the superior part. Petechial haemorrhages present throughout the organ. The pancreatic duct was filled for about six inches above the outlet with a firm, caseous material, which when removed retained its shape. Some redness noted, but no definite swelling of the intestinal mucous membrane in region of the diverticulum.

Case V.

This animal was a grade gelding, nine years of age and weighing approximately 1200 pounds. A year before, he had gone through an attack of influenza making uninterrupted recovery. The appetite was poor for about a week before the commencement of the illness about to be described.

The onset was very sudden. Rigors and abdominal pain ushered in the acute attack. The animal was examined a few hours later, and found to have a temperature of *105.8°F.*, pulse *120*, and respirations *68;* and the skin bathed in perspiration.

Physical Examination

1. *Head.*

The mucous membrane of eyes, icteric. No nasal discharge present, but mucous membranes stained faintly yellow.

Mouth and tongue, slightly icteric, and a thick, tenacious mucus present.

2. *Chest.*

Respiratory movement on left and right side, even.

Auscultation and percussion failed to disclose pneumonia or pleurisy.

3. *Abdomen.*

Animal evidenced pain, but no rigidity, in the abdomen region. The bowel did not appear overloaded or distended with gas. The faeces were normal in appearance.

4. *Legs and Extremities.*

No laminitis present; the corresponding legs being equal in size, with no enlargement of the lymphatics.

Emaciation was very rapid. The faeces retained a normal colour throughout, although slight jaundice was present during the first few days of illness. The course was progressively downwards, and sixteen days after first examination the animal became extremely weak and refused all food. Three days later he died.

Necropsy

The pancreas was found enlarged, with many haemorrhagic areas about the size of a bean throughout the structure. Necrosis was present in the lower part, the duct of Wirsung being filled with material which appeared to be partly made up of tissue shreds. The size and colour of the liver was normal with no enlargement or occlusion of the duct.

For the purpose of clarity, the principal data contained in the case reports is compiled in Table I.

Case	Breed	Age	Sex	Weeks ill before acute attack	Abdominal pain	Jaundice	Laminitis	Lymphagitis	Days before death following acute attack
I	Percheron	5	M	3	Yes	Yes	Yes	No	20
II	Clydesdale	7	M	3	Yes	Yes	Yes	No	6
III	Belgian	6	M	5	Yes	Yes	Yes	No	9
IV	Grade	9	F	12	Yes	Yes	No	Yes	8
V	Grade	9	M	1	Yes	Slight	No	No	19

DISCUSSION

The lack of laboratory work in connection with these cases is regrettable. Thus the necessity is self-evident for determining the number of diastase units contained in the urine, also the presence of bile. Likewise the postmortem examinations should have been supplemented by bacteriological and histological examinations of the various tissues involved. The extenuating circumstances are, however, that these cases occurred in a general practice covering a wide territory and that the observations were recorded in a case book kept for the author's information, with no thought of publication. The records were shown to a colleague who insisted that, irrespective of their deficiencies, the cases in question should be placed on record.

Therefore, discussion must be based on data which, unfortunately, lacks supplemental laboratory evidence which might be useful in giving a clearer conception of the disease in question.

It will be first noted that death occurred in all the animals referred to in the report. Is a fatal ending to be expected in all cases of pancreatitis? An answer to this is not possible since only cases have been dealt with which came to necropsy, and in which pancreatitis was demonstrated. Other horses have been under observation which clinically bore a resemblance to those considered, but in general they did not evince the prolonged temperature or the same degree of shock. It is very possible, therefore, that these animals had a cholangitis with no involvement of the pancreas or its duct. A study of future cases, with the diastase content of the urine being given consideration, may assist to a better understanding.

It will be noted that four of the five infected animals were males. In the district in which these cases were found there is a great preponderance of females. It would therefore seem that the disease occurs more often in males.

An interesting feature is the relatively long period during which four of the animals showed a slight abdominal disturbance. It will be noted that this ranged from three to twelve weeks, during which time they were treated with domestic remedies. It was only when the symptoms became grave that assistance was sought. Therefore the length of time illness was present before assistance was called may be looked upon as the period of prodromal symptoms ushering in the acute attack. With one exception, this was from three to twelve weeks. A better understanding of the pathology causing these initial symptoms would doubtless have much significance. Is pancreatitis present during this period or is a low grade intestinal infection present which later involves the duct of Wirsung, causing a complete or partial occlusion, thus bringing about a condition favorable for the activation of the trypsin within the gland?

It will be noted that jaundice was present in all animals. This might be taken to suggest that an initial infection commenced in the bowel in the region of the diverticulum duodeni causing occlusion of the bowel and pancreatic ducts. The histories however do not indicate that jaundice was present during the prodromal period. Therefore the early symptoms must have resulted from a local intestinal infection involving the diverticulum duodeni with a later occlusion of the bile and pancreatic ducts, or the pancreatitis was the first pathological change, and was followed by a low grade infection of the ducts. Since no bacteriological study was made it may be surmised that the local tissue changes were brought about by a streptococcic or colon bacillus invasion. There is, however, no supporting evidence of this view.

Treatment

Until more is known about pancreatitis of the horse and more especially the contributory causes, little can be done in a way of specific treatment.

If the pancreatitis is the initial affection then little assistance can be gained by therapeutic measures, and no doubt the disease will run a fatal

course. If however, the disease results from an initial intestinal infection, then some assistance may be given. It is advisable to deal with cases with the latter conception in view, since it is the only alternative that offers encouragement.

Mild saline laxatives should be given to unload the bowel after which time the bowel wall should be given the greatest possible rest. Easily digested food should be sparingly administered once or twice a day, the object being to reduce peristalsis to a minimum. The body fluid should be conserved by administering, intravenously, at least a gallon of normal saline each day.

Bismuth carbonate and Sodium salicylate in requisite doses may prove useful, and sulphanilamide compounds should be tried, having regard to the possibility of a local streptococci invasion.

REFERENCES

1. Osler's Principles and Practice of Medicine. Appleton. Eleventh Edition.
2. Rich, Arnold R. and Duff, G. Lyman. Bulletin of the Johns Hopkins Hospital. 1936. Vol. LVIII, No. 3, pp. 137-211.

Cyclopropane

An Improved Anaesthetic Agent

CYCLOPROPANE, a saturated hydrocarbon, discovered by Henderson and Lucas in 1928 at Toronto, was first applied clinically by Dr. Waters of Wisconsin University. It was introduced into Canada by Dr. H. R. Griffith* and has been used by them in over 2500 cases; he feels it is safe, dependable, controllable, and almost universally applicable. It is heavier than air and is marketed as a compressed gas in cylinders. It produces narcosis when inhaled in a concentration as low as 4% but 10%—15% is the average concentration for deep surgical anaesthesia. It is economical, costing 30 cents a gallon and only one and a half gallons are required for one hour surgical anaesthesia when the CO_2 absorption technique is employed. Experiments show that it has no effect on the kidney or liver, that there are little, if any, metabolic upsets and that the pH and CO_2 combining power of he blood are more normal than with other anaesthetics: the excess of oxygen which is present at all times is, no doubt, partially responsible for such favourable findings. In too high concentration it causes respiratory failure and cardiac collapse. Griffith has used it in almost every type of patient (ages 7 weeks to 90 yrs.) and for all kinds of operations. Induction which takes 3—4 minutes and is more pleasant than nitrous oxide, does not cause respiratory irritation. The adequate muscular relaxation obtainable for abdominal surgery and the ease of endotracheal administration for head and neck operations make cyclopropane particularly desirable. As regards the objections that bleeding is more profuse and that there is danger of explosions with cyclopropane, Griffith points out that the former is a disputed point and that the danger of explosion is no greater with cyclopropane than with ether, ethylene or nitrous oxide.

*Can. Med. Assoc. Journal, May 1937, Vol. 36, No. 5.